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Role of circulating placenta-derived extracellular vesicles in preeclampsia

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Objective

To investigate the contribution of placenta-derived extracellular vesicles (EVs) in preeclampsia associated endothelial dysfunction.

Methods

Sera samples from 8 preeclamptic and 6 normotensive pregnancies were pooled into 3 preeclampsia pools and 1 control pool. Placenta-derived EVs were isolated by differential centrifugation. To assess endothelial damage, endothelial cells in culture were incubated for 48h with preeclampsia or control sera in the presence or absence of EVs (depleted sera). Then, changes in endothelial dysfunction markers (VCAM-1, ICAM-1), von Willebrand factor (VWF) expression and reactive oxygen species (ROS) production were assessed. The results were expressed as the average fold increase versus control.

Results

VCAM-1, VWF and ROS were significantly higher in cells exposed to preeclampsia sera versus control sera [fold change of 5.4, 3, and 1.3, respectively, $p<0.05$]. VCAM-1, VWF and ROS were significantly lower in depleted preeclampsia sera compared to non-depleted sera [fold change of 5 vs 0.7 for VCAM-1, 4.3 vs 2.8 for VWF, and 1.3 vs 1.2 for ROS, $p<0.05$]. The cell exposure to control sera supplemented by placenta-derived EVs from preeclampsia increased significantly VCAM-1, VWF and ROS compared to non-supplemented control sera [fold change 3.9, 4.1 and 1.4, respectively, $p<0.05$].

Conclusion

Circulating placenta-derived EVs are potentially pathogenic for the endothelial damage associated with preeclampsia since they trigger an oxidative, prothrombotic and proinflammatory state.